Community-Associated Methicillin Resistant Staphylococcal Aureus in Children

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Case Study 2
Subjective

Case Selection

**Reason for selecting case.** I chose this case because of my interest in antibiotic resistant infections. I was aware that hospital-associated methicillin resistant staphylococcal aureus (HA-MRSA) is an enormous fiscal and healthcare burden in the United States. I was not familiar with the issues surrounding the more recently emerging community-associated methicillin resistant staphylococcal aureus (CA-MRSA). A case study is a perfect venue to explore this issue in populations that have not traditionally been impacted by MRSA—children living in the community.

**Site.** Ennis Children’s Clinic, a pediatric clinic located in the small community of Ennis, Texas. It is located about 1 hour south of Dallas, Texas. This clinic is designated a rural health clinic.

**Patient profile.** A.S. is an 8-year-old White male who has been a patient of this clinic since his birth. His siblings are also patients here.

**Encounters.** I had one encounter with this patient and his mother for this issue.

**Insurance.** The patient is enrolled in Medicaid insurance. His plan is administered through the Parkland Health Plus Plan.

History

**Chief complaint.** A.S. states, “My knee hurts.”

**History of presenting illness.** His mother brought A.S. to the clinic this morning. She reports that yesterday, while riding his bike, he fell off and hit his left knee. This morning when he woke up he complained of pain in that knee. She saw that the knee was swollen and brought him to the clinic instead of sending him to school. A.S. thinks his knee “looks green.”
**Past medical history.** A.S. was a spontaneous vaginal delivery without complications. His EGA was 40 weeks and his birth weight was 7 lbs 2.3 oz. His past medical history was positive for five episodes of acute otitis media, bronchitis, three episodes of bronchiolitis, chickenpox, conjunctivitis, fracture of left 5th digit of foot, influenza, sinusitis, a supraorbital laceration, thrush, two episodes of tonsillitis, and urticaria. Ongoing issues include allergic rhinitis, an elevated body mass index, failed vision test, and phimosis.

A.S. has had three soft tissue infections since birth (they are listed separately for emphasis). At the age of four months, a right great toe infection was treated with Bactroban three times daily. At the age of four years and 11 months, a pustule and cellulitis of the right foot was treated with Reflex 500 mg twice a day for 10 days. At the age of seven years and 10 months, an abscess was treated with Bactroban and Bactrim.

Currently, A.S. is on no medications. He has no known drug allergies. His immunizations are complete and up-to-date.

**Family history.** The patient’s mother has a history of CA-MRSA, but no further details are available.

**Social history.** A.S. lives with his mother, father, and three siblings. He is in first grade. He has had difficulty learning to read and has a tutor for assistance.

**Review of systems.**

**General.** No fever or chills; normal sleep, bowel, and bladder.

**ENT.** He has had nasal congestion and a runny nose for the past 2 weeks.

**Cardiology.** No chest pain or shortness of breath.

**Pulmonary.** He has had a non-productive cough at night for the past 3 weeks.

**GI.** Appetite is normal. There is no abdominal pain, nausea, vomiting, or diarrhea.
Musculoskeletal. He is able to fully bend the affected left knee. He is able to weight bear. There is no hip or ankle pain.

Skin. No rashes or bruising.

A Brief Discussion of Pathophysiology

Soft tissue injuries of the knee are common disorders in patients presenting for emergency care. It is estimated that there are more than 1 million emergency department visits and 1.9 million primary care outpatient visits yearly for acute knee pain (Smith, Stahel, Morgan, & Trafton, 2008). It is important to establish clear diagnoses and therapeutic objectives for these injuries (Smith et al., 2008).

Knee pain and related symptoms can be caused by damage to the ligaments, muscles, tendons, and menisci; from infection to the knee joint or surrounding structures; or from trauma to the bones forming the joint (Smith et al., 2008). Part of the assessment includes determining if the acuity is due to trauma, infection, or exacerbation of a chronic condition. For most patients, the diagnosis can be determined from a targeted history, focused physical examination, and the workup which can include diagnostic imaging (e.g., plain radiography) (Smith et al., 2008).

Objective

Physical Exam Data

General. Patient is alert, well nourished, and responsive to questions.

HEENT. Normocephalic, no lesions. Clear sclera. Pupils equal and reactive to light and accommodation. There is no nystagmus. Nares are patent, and here is clear nasal discharge. Tympanic membranes are pearly gray and intact bilaterally. Throat is clear and tonsils are not enlarged.

Neck. Supple, no lymphadenopathy.
Cor. Rate and rhythm regular. No murmurs or extra sounds.

Chest. Lungs clear to auscultation, normal shape and expansion, no wheezes, rhonchi, or rales.

Abdomen. Bowel sounds present all quadrants, normoactive, soft, non-distended, nontender, no organomegaly.

Musculoskeletal. Left knee landmarks were distorted due to swelling. Range of motion of the knee was knee is full. Muscle strength is intact. Ballottement is negative. Valgus and varus maneuvers are intact, and drawer signs are negative. The range of motion of left hip and ankle were within normal limits.

Skin. Is well perfused. There is generalized tenderness, erythema and swelling over the left patella. There is a warm fluctuant mass, about 1.5 by 2cm over the superior portion of the patella. A lighted magnifying glass confirmed a yellowish tinge.

Scientific Underpinnings of Data

Physical examination of the knee consists of inspection, palpation of the superficial anatomy of the patellar and the patellar region, assessment of range of motion and strength, assessment for patellar effusion, and evaluation for ligamental instability and damage to the menisci (Physical Exam of the Knee, n.d.). When assessing orthopedic injuries in children, it is important to remember that children have a much higher incidence of fracture versus sprain than adults do. A fracture should be ruled out in any child who has significant pain at the site of injury, or if he refuses to walk on the extremity (Crawford, 2003). If the physical exam findings of pain, erythema, swelling, and abscess are consistent with infection, the type of organism needs to tentatively be identified so that appropriate antibiotic therapy can be initiated (Bacterial Skin Infections, 2006). The most common organisms to cause skin infections are staphylococcal...
aureus (S. aureus) and Group a beta hemolytic streptococcus. The type of infection can often be identified by appearance: streptococcal infections cause diffuse, rapidly spreading infection while staphylococcal cellulitis is more localized and usually occurs with an open wound or abscess (Bacterial Skin Infections, 2006).

Assessment

Differential Diagnoses

Presenting problem and differential diagnoses. The patient’s presenting problems are staphylococcal cellulitis and abscess of knee (ICD-9 296.34) and methicillin resistant staphylococcus aureus (ICD-9 041.12). The differential diagnoses are streptococcal infection, soft tissue injury, and patellar fracture.

Assessment of the presenting complaint. The physical exam excluded ligamental and meniscus injury as the source of pain. A patellar fracture was considered, but the patient was able to ambulate and did not appear to be in significant pain. The appearance of the injury, especially presence of an abscess, was consistent with a staphylococcal infection. If the pain persisted after resolution of the infection, a radiograph of the knee could be considered. The infection was presumed to be CA-MRSA because of his mother’s positive history of CA-MRSA, and because the patient had an infection 1 month ago with similar features. This clinical practice does not routinely culture abscesses.

Discussion of staphylococcal aureus infections. Staphylococcal aureus (S. aureus) is a gram positive, catalase positive bacteria. Of the 17 different species of staphylococcus that cause infection in humans, it is the only species that is coagulase positive. Microscopically, it has the appearance of grape clusters. S. aureus is very hardy and can survive extreme conditions of drying, heat, low-oxygen and high-salt environments. It has many surface proteins that allow the bacteria to bind to tissues and foreign bodies (Committee on Infectious
S. aureus is responsible for many localized and invasive infections, as well as three toxin-mediated syndromes: toxic shock syndrome, scalded skin syndrome, and food poisoning. The localized infections include hordeolum, furuncles, carbuncles, impetigo, paronychia, eczema, cellulitis, omphalitis, parotitis, lymphadenitis, and wound infections (Committee on Infectious Diseases, 2009). S. aureus also causes infections associated with foreign bodies, including catheters, grafts, pacemakers, peritoneal catheters, cerebrospinal fluid shunts, and prosthetic joints. Bacteremia can be complicated by septicemia, endocarditis, pericarditis, pneumonia, pleural empyema, soft tissue, muscle, or organ abscesses, arthritis, osteomyelitis, and septic thrombophlebitis of large vessels (Committee on Infectious Diseases, 2009). S. aureus aspiration pneumonia also can occur in the setting of mechanical ventilation or influenza. Risk factors for severe infections include chronic diseases, such as diabetes mellitus and cirrhosis, immunodeficiency, nutritional disorders, surgery, and transplantation (Committee on Infectious Diseases, 2009).

In community settings, S. aureus is transmitted most often by direct contact. In healthcare settings, it is spread patient to patient via the hands of healthcare professionals (David & Daum, 2010). Other modes of transmission are via colonized healthcare professionals and family members (usually of the nares or skin), or fomites such as contaminated surfaces and objects. Because S. aureus can colonize the nares, increased dissemination occurs during viral upper respiratory tract infections (David & Daum, 2010).

At the onset of the era of antibiotics, S. aureus was susceptible to beta lactam antibiotics such as the penicillins and cephalosporins. The widespread exposure to these classes of drugs induced adaptive mechanisms in these bacteria. First, they responded by producing beta lactamase, an enzyme that is capable of break beta lactam rings. This rendered many drugs in
this class of antibiotics useless, especially the penicillins. The second adaption consisted of moving the penicillin-binding site (PBS) (the place on the bacterial cell membrane where the antibiotic became bound). As a result, the bacteria no longer “recognize” the antibiotic. This mechanism accounts for the development of MRSA in these organisms (Staph Aureus, 2010). In the 1980s, hospitals in the United States became inundated with a new type of S. aureus infection (David & Daum, 2010). These bacteria were resistant to the antibiotics commonly used to treat staph infections. HA-MRSA, as it became known, was confined largely to hospitals and healthcare environments. The risk factors for acquiring HA-MRSA were (a) residence in a long-term care facility or acute-rehabilitation unit; (b) indwelling line or catheters; (c) surgical wounds; (d) chronic liver, lung, or vascular disease; (e) malignancy; (f) recent exposure to antibiotics; (g) intravenous drug use; (h) ICU admission; or (i) exposure to a patient with any of these risk factors for MRSA (David & Daum, 2010). HA-MRSA continues to be a significant and costly source of morbidity and mortality in hospitals today. In the 1990s another form of MRSA emerged. These infections were more likely to be of the skin and soft tissues (SSTIs), although necrotizing pneumonia and severe sepsis could occur. CA-MRSA affected populations not traditionally at risk for MRSA infections, including populations of correctional facilities, military training facilities, childcare centers, and members of sports teams. CA-MRSA has also occurred in people without direct links to those settings. David and Daum (2010) discussed the differences in genetic and antibiotic resistant patterns of the two isolates as they relate to type of gene cassette (a DNA sequence encoding one or more genes for a single biochemical function. (Gene Cassettes, 2010,) and the presence of the Panton-Valentine leukocidin (PVL) gene. PVL is a cytotoxin that can destroy white blood cells and cause extensive tissue necrosis and severe infection (Panton-Valentine Leukocidin, 2010). A summary of their differences is found in Table
1. One definition of CA-MRSA is “Infections occurring among outpatients or among inpatients with an MRSA isolate obtained earlier than 48 hours after hospitalization” (David & Daum, 2010, p. 619).
Table 1

*Differences between CA-MRSA and HA-MRSA*

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>CA-MRSA</th>
<th>HA-MRSA</th>
</tr>
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<tbody>
<tr>
<td>Type of Staphylococcal</td>
<td>Smaller SCCmec</td>
<td>Large SCCmec</td>
</tr>
<tr>
<td>chromosomal elements</td>
<td>elements, usually</td>
<td>elements type I, II, or III</td>
</tr>
<tr>
<td>cassette</td>
<td>SCCmec type IV or III</td>
<td>type V</td>
</tr>
<tr>
<td>mec (SCCmec).(^a)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>PVL gene.b</td>
<td>Frequently carries</td>
<td>Rarely carries</td>
</tr>
<tr>
<td>Resistant to many</td>
<td>Less often</td>
<td>Often</td>
</tr>
<tr>
<td>classes of non-β-lactam antimicrobials</td>
<td></td>
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The Committee on Infectious Diseases (2009) endorsed the following algorithm (Figure 1) to guide the treatment of CA-MRSA infections.
Figure 1. Algorithm for Initial Management of Skin and Soft Tissue Infections Caused by Community-Acquired Staphylococcus Aureus

Figure 1. Adapted from “Staphylococcal Infections” by Committee on Infectious Diseases. (2009). In L. K. Pickering (Ed.), Redbook 2009 Report of the Committee on Infectious Diseases (28th ed.).
Plan with Rationale

1. Mupirocin 2% ointment: apply to affected area small amount externally twice a day for 7 days.
   

2. Trimethoprim-Sulfamethoxazole 400-80 mg tablet: take 2 tablets orally twice a day for 7 days.
   
   Rationale: MRSA is resistant to trimethoprim or sulfamethoxazole alone, but may be susceptible to the combination. The antimicrobial activity of the combination is due to its actions on two steps of the enzymatic pathway for the synthesis of tetrahydrofolic acid (Petri, 2009).

3. Warm soaks three times a day to promote drainage (Bacterial skin infections, 2006).

4. Incision and drainage was considered per recommendation of algorithm (see p. 11), but constraints unique to this practice precluded this procedure.

5. Refer to the emergency room if fever develops or if there is no improvement within 48 hours.

Continuity of Care

Outcome of Interventions

Follow-up. I was unable to contact the family for follow-up. If the patient had not improved, he would have been referred for an incision and drainage at the local emergency room.

Referrals. Consultation with an infectious disease specialist to reflect on eradication strategies was considered but not able to be completed. The only practice that accepts the patient’s insurance has very strict referral criteria, which the patient did not meet.
Rationale for referral: *S. aureus* colonizes the skin and mucous membranes of 30% to 50% of healthy adults and children. The anterior nares, throat, maxilla, perineum, vagina, or rectum are usual sites of colonization (Committee on Infectious Diseases, 2009). Recurrent skin infections should raise suspicion of colonization, such as staphylococcal nasal carriage (Bacterial skin infections, 2006). There is insufficient evidence to support use of topical or systemic antimicrobial therapy for eradicating nasal or extra-nasal MRSA (Loeb, Main, Easy, & Walker-Dilks, 2003; Gorwitz, Jernigan, Powers, & Jernigan, 2006). After treating infection and reinforcing hygiene and appropriate wound care, consider consultation with an infectious disease specialist regarding use of decolonization when there are recurrent infections in an individual patient or members of a household (Centers for Disease Control and Prevention [CDC], 2007).

Family and Patient Education

1. Finish all antibiotics as prescribed.

2. This infection is easily passed from person to person. To prevent other family members from becoming infected, keep draining wounds covered.

3. Seal dirty bandages in a plastic bag before disposing of them.

4. Wash sheets, towels, and clothes with hot water and laundry detergent and dry them in a hot dryer.

5. The patient and family members should wash their hands with soap and water or an alcohol based hand sanitizer, and clean under fingernails as well.
6. It is especially important to wash hands after touching the wound or changing the bandage.

7. Avoid sharing personal items like towels, washcloths, razors, clothing, athletic equipment, or uniforms.

(Adapted from Community-Associated Antibiotic-Resistant Staph Infection [CA-MRSA], 2009).

If any of the children in this family play sports, they should

1. Shower after working out at a gym or on the playing field.

2. If athletic equipment is shared, use a barrier between skin and equipment

3. Use the same equipment with each practice rather than sharing, if possible. The surfaces of the equipment should be cleaned between each use.

(Adapted from Community-Associated Antibiotic-Resistant Staph Infection [CA-MRSA], 2009).

Addendum: At the time of this writing, one of the patient’s siblings had presented for treatment of an abscess, which was presumed to be CA-MRSA.
References


